U.S. Department of Health and Human Services
NIH News
National Institutes of Health

National Institute on Drug Abuse
http://www.drugabuse.gov/

EMBARGOED FOR RELEASE
March 28, 2010
1:00 p.m. (EST)

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COMMON MECHANISMS OF DRUG ABUSE AND OBESITY

Research Suggests Food Availability Could Prompt Addiction

Some of the same brain mechanisms that fuel drug addiction in humans accompany the emergence of compulsive eating behaviors and the development of obesity in animals, according to research funded by the National Institute on Drug Abuse (NIDA), a component of the National Institutes of Health.

The study, conducted by researchers at the Scripps Research Institute, was released today in the online version of Nature Neuroscience and will also appear in the journal's May 2010 print issue. When investigators gave rats access to varying levels of high-fat foods, they found unrestricted availability alone can trigger addiction-like responses in the brain, leading to compulsive eating behaviors and the onset of obesity.

"Drug addiction and obesity are two of the most challenging health problems in the United States," said Dr. Nora D. Volkow, director of NIDA. "This research opens the door for us to apply some of the knowledge we have gathered about drug addiction to the study of overeating and obesity."

Both obesity and drug addiction have been linked to a dysfunction in the brain's reward system. In both cases overconsumption can trigger a gradual increase in the reward threshold—requiring more and more palatable high fat food or reinforcing drug to satisfy the craving over time.

Researchers conducted this study in three groups of male rats over a 40-day period. Each day, the three groups had unlimited access to standard lab food. In addition, two of the groups also had access to high-fat, cafeteria style foods for short (one-hour) or long (18-23 hours) periods.

After 40 days, all groups were denied access to the high-fat foods. Throughout the study, researchers observed the feeding behaviors of each group, noting caloric intake, weight gain, and brain response.

The results support the notion that type 2 dopamine receptors (D2DR)—brain receptors that have been shown to play a key role in addiction—also play a key role in the rats' heightened response to food. In fact, as the rats became obese, the levels of D2DR in the brain's reward circuit decreased. This drop in D2DR is similar to that previously seen in humans addicted to drugs like cocaine or heroin.

"The results of this study could provide insight into a mechanism for obesity," said Paul J. Kenny, one of the study's co-authors and an associate professor at the Scripps Jupiter, Fla., research facility. "It's possible that drugs developed to treat addiction may also benefit people who are habitual overeaters."

Study results also suggest that environmental factors, such as increased or unlimited access to high-fat food options, can contribute to the problem of obesity.

"Hopefully, this study will change the way people think about eating," said Paul Johnson, a co-author and graduate student in the department of molecular therapeutics. "It demonstrates how just the availability of food can trigger overconsumption and obesity."

The study titled: "Addiction-like reward dysfunction and compulsive eating in obese rates: Role for dopamine D2 receptors," by Paul M. Johnson and Paul J. Kenny in Nature Neuroscience can be found online at:
http://www.nature.com/neuro/journal/vaop/ncurrent/index.html

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